Scientific and Regulatory Aspects of Venezuelan Equine Encephalitis[1]

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ABSTRACT

Recognized mosquito-borne, viral encephalomyelitis in the United States consists of Western (WEE), Eastern (EEE), Venezuelan (VEE), St. Louis (SLE), and California (CE) strains. WEE, EEE, and VEE cause clinical disease problems in equines. Horses are dead-end hosts for WEE and EEE but are amplifying hosts for VEE. Thus, the latter is of greater public health significance. Laboratory support is required for differential diagnosis of the three diseases. Equines are considered primary sentinels for the detection of epizootics of VEE.

VEE swept across Mexico, in less than 1 year, killing in excess of 10,000 horses. Epidemic VEE entered the United States from Mexico in June 1971, and appears to have established itself in the small mammal populations of Texas. Numerous mosquito species in the United States are capable of transmitting VEE. The disease was brought under control using aerial pesticide spraying, quarantine, and vaccination of horses. The epidemic virus of VEE has been isolated from animals in 25 Texas counties. It has not been found outside Texas.

Equine encephalitis has been with us for a considerable time. An epizootic of what was probably viral encephalitis of horses occurred on Long Island, New York, in 1828 and again in 1836 and more than 500 horses died. In 1882 and 1897, horses in Texas died in numbers described as "by the thousands." In 1912, a disease called Horse Plague caused losses estimated at 35,000 in Nebraska, Kansas, Colorado, Oklahoma, and Missouri. In the 1930's, equine encephalitis was regarded as the most important disease affecting horses in the United States. During the 1930's, much additional information was developed in regard to the encephalitides of Equidae. The eastern type (EEE) and the western type (WEE) of equine encephalitis were found to be caused by filterable viruses, and in South America a virus (VEE) was found to be causing a similar encephalitis of horses.

Early clinical signs of encephalomyelitis in horses include marked depression and high fever. Other signs include incoordination, circling, mystagmus, drooping lower lip, and dehydration. The disease may be acute or subacute and may result in prostration and death, or incomplete or complete recovery. In an outbreak situation many infected horses may not show clinical evidence of the disease.

WEE virus was isolated from horses in California in 1930. Human cases were first described in 1932 and the virus first isolated

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from man in 1938. The virus of WEE was isolated from mosquitoes in 1941. WEE is found primarily in western and midwestern states and also in western Canada where it has extended to as far north as the south part of Hudson Bay. The disease has also been reported in some southeastern states of this country and WEE virus has been recovered from birds and mosquitoes in northeastern United States. WEE probably also exists in South America in Argentina, Peru and Chile. *Culex tarsalis* mosquitoes (which feed on birds, horses, and man) are considered the primary vector of WEE with wild birds a primary reservoir. Domestic birds can also become infected and WEE virus has also been isolated from squirrels and garter snakes. Significant outbreaks of WEE occur in man as well as horses, both horse and man can be considered dead-end hosts of the virus. The case-mortality rate in horses is about 50%.

EEE virus was isolated in 1933 following investigation of equine encephalitis in New Jersey, Virginia, Delaware, and Maryland. The virus was isolated from humans, pigeons and pheasants in 1938. EEE virus has been found in Canada, eastern and southern United States, and along the gulf coast area extending through Mexico and Central America, and northern and eastern South America as far south as Argentina. Islands in the Caribbean have been involved. The epizootiology of EEE includes a sylvan cycle involving fresh-water swamp mosquitoes and swamp-dwelling birds. The virus can thus cycle over a long period of time until the opportunity is presented for it to "spill over" to other birds and to other mosquitoes which attack horse and man. Horse and man are considered, as with WEE, for practical purposes, to be dead-end hosts. EEE virus causes significant disease outbreaks in horses but fewer cases in man than does WEE. In pheasant flocks direct bird-to-bird transmission has been found.

St. Louis encephalitis (SLE) was recognized in the early 1930's and since has been an important largely urban and sometimes rural mosquito-borne human disease of the continental United States, northern Mexico and the Caribbean. The virus can cycle between mosquitoes and wild birds with man as incidental dead-end host. SLE is not important as a disease of horses nor are they or other mammals, including man, believed to perpetuate the disease. Horses do develop serological SLE titers.

California encephalitis virus (CE) was first isolated from mosquitoes in California in the early 1940's and since has been found in widely separated states. Small wild mammals (perhaps rabbits and squirrels) and mosquito vectors probably maintain the virus in nature. Horses are not an important factor insofar as CEV is concerned.

When one recalls that there are more than 200 recognized arboviruses, I suppose it is only natural that this array can create rather difficult and sometimes bewildering problems. The few just mentioned are those which may come to the attention of scientists in this country. The encephalitides are important diseases of horses and man and from both an economic and public health standpoint are cause for considerable concern to the livestock owners, the general public, and to public health, research and regulatory scientists.

Venezuelan Equine Encephalomyelitis (VEE)

VEE was first noted in horses in Colombia in 1935 and in Venezuela in 1936. The virus was first isolated from an equine brain in Venezuela and identified in 1939. VEE appeared as epizootics from time to time in these countries and was later reported in Trinidad, Ecuador, British Guiana, French Guiana, Surinam, Brazil, Curacao, West Indies, Peru, and Panama. The disease has been observed to cause significant outbreaks in humans since 1944. In 1961 and 1962, a severe outbreak of the disease occurred in humans in Colombia, northwest Venezuela (32,000 cases were reported with at least 190 fatalities) and in Panama where several hundred people became infected. In Ecuador, 31,000 human cases with 250-400 deaths were reported in 1969.

VEE Virus

Like other arboviruses of Group A, VEE is a relatively small (40-70 mu) RNA virus. It

Fig. 1.–Colt with VEE symptoms, Brownsville, Tex.

is not readily inactivated by formalin and can be preserved by lyphilization or in 50% buffered glycerol at -70°C. It can be readily recovered from blood and nasopharyngeal washings if collected during the acute phases of the disease and for longer periods of time from other tissues (e.g., bone marrow, spleen, liver, lung, kidney, thymus, adrenal, heart, lymph nodes). The virus has been isolated from the nasal, eye and mouth secretions and from the urine and from milk of infected mares. VEE virus can be inactivated by propylene glycol, glycolic acid, thioglycolic acid, thiourea, and methyl thioglycolate.

This agent is notorious for its ability to infect laboratory personnel working with the virus, usually by inhalation of airborne material. No chemical has been shown to have significant activity, in vivo, against VEE virus, thus specific chemotherapy is not available at present. This also explains the desirability for a vaccine as was developed by the Army in their TC-83 vaccine for protection of high-risk personnel.

Horses affected with VEE may die of either a fulminating systemic disease or a typical encephalitis. It is not possible to distinguish clinically among the diseases referred to as WEE, EEE, and VEE. Histologic changes furnish only presumptive evidence. Unfortunately, a veterinarian is often called to examine a suspect case after obvious clinical signs are present and viremia is decreasing and antibody levels for serological tests have not yet peaked.

We must depend upon the laboratory for serological and virus isolation work in order to determine which virus is involved.

Three serological tests are used routinely. Listed in the order of their specificity they are:

1. (SN) Virus-Serum Neutralization Test: Neutralizing antibodies often appear within a few hours after infection and may persist throughout the animal's life.

2. (CF) Complement-Fixation Test: At higher antibody levels the CF test is apparently quite specific for VEE.

3. (HI) Hemagglutination Inhibition Test: Using the HI test, a relatively high degree of cross reactivity between VEE and other virus has been observed. Antibodies detected by HI appear somewhat later and probably persist throughout the host's life.

Significant SN, CF, and HI antibodies may develop as early as six days after virus inoculation. Virus isolations may be made by using a variety of laboratory animals including mice, rats, guinea pigs, hamsters, and monkeys. Embryonated hens' eggs readily become infected and die in 15-48 hours.

Chicken embryo, fetal guinea pig heart, baby hamster or bone marrow cells can also be used as tissue culture systems. A practical commonly used laboratory procedure is to inoculate suckling mice with tissues from the suspect case (blood, serum, spleen, brain).

If mice are killed, a crude complement fixation antigen is made from the mouse brain. If the CF test is positive for VEE, the mouse brain is inoculated into guinea pigs or adult mice. If these are killed, this is taken as evidence that epidemic VEE virus is present. If they do not die, this is evidence that either vaccine virus or an endemic strain of VEE is involved. The agent may be further identified by means of the kinetic HI test.

VEE in Man

The incubation period in man is considered to be short, ranging from 2-5 days; the onset is usually very sudden. Symptoms may include severe headache, fever lasting from 1-4 days, malaise, chills, nausea or vomiting, and myalgia. Severe encephalitis or generalized systemic illness may occur, and in rare instances, tremors, diplopia, and lethargy. Symptoms persist 3-5 days in mild cases and as long as 8 days in more serious attacks. A prompt and apparently complete recovery usually takes place. Fatalities generally involve children less than 15 years of age.

VEE Distribution in Wild Mammals and Birds

Isolation of VEE virus from wild mammals includes those from monkeys, fox, spiny rat, forest spiny pocket mouse, terrestrial rice rat, short-tailed cane mouse, cotton rats, opossums, and from certain species of wild birds. Bats appear experimentally to be excellent hosts for the VEE agent. Significant viral titers persisted for at least 26 days and low titers for at least 90 days. The infection is apparently not lethal to the bats.

The VEE virus differs from EEE and WEE viruses in that it seems to multiply better in mammals than in birds. However, VEE virus has been isolated from a number of naturally infected pigeons, chickens, sandwich tern, green heron, groove-billed ani, little blue heron, black-cowled oriole, gray-capped flycatcher, social flycatcher, keel-billed toucan, scarlet-rumped tanager, and clay-colored robin. The 10 last-named species of birds were caught in Panama, although several of these migrate to North America.

English sparrow-mosquito-English sparrow transmission was demonstrated experimentally. Virus levels were usually quite low in all birds inoculated or subjected to mosquito bites, and the birds did not show clinical signs or encephalitis. One species of striated herons with rather low viremias were able to infect feeding mosquitoes. Chicks less than one month old were fatally susceptible to experimental infection; those older than one month produced antibodies but no clinical signs or viremia.

VEE Distribution in Domestic Animals

VEE virus has been isolated from naturally infected horses, mules, and donkeys. Neutralizing and HI antibodies of significant titers have been found in sera from dogs, goats, swine, sheep, and cattle. Of dogs experimentally inoculated with VEE, some died of the infection. Contact transmission from infected to noninfected dogs was demonstrated.

Epizootiology of VEE

The epizootiology of VEE seems to include a sylvan cycle utilizing mosquitoes and wild rodents such as the cotton rat. A large number of species of mammals and birds develop antibodies to VEE virus and virus can be isolated from many of these. Many different species of mosquitoes can be vectors of the disease; however, depending on geographical and climatical factors, and the strain of VEE virus involved. Some species are more efficient vectors than others. Although the mosquito has not been proven to transmit the virus transovarally, the virus multiplies considerably in the mosquito, which can remain infected for life.

Viremia in the horse persists only for a few days. However, it can reach such high levels as to infect nearly all appropriate species of mosquitoes which feed upon it. Contrary to the other viral encephalitides we are

concerned with, the horse serves as a very important amplifying host, thus is extremely important from a public health standpoint. One often hears the expression, perhaps an oversimplification, that if you don't have sick horses you don't have an epidemic in humans. However, man can also develop a rather high viremia. Although direct transmission from horse to horse has been experimentally proved, it is unlikely that this plays a significant part in the natural spread of the disease.

In order to understand the encephalitides of man and animals, it is essential that we develop the best possible information on the viral agents involved, which vectors and which host animals are instrumental in maintaining the disease in endemic or sylvatic form, and those hosts necessary for the development of epidemics. It is necessary to determine virus-vector-host relationships, which vectors are potentially the most efficient as well as which animals are capable of infecting them. Many warm or cold-blooded animals are susceptible to mosquito-borne infection, but only certain ones develop sufficient levels of viremia to infect mosquitoes with minimal infection thresholds.

To be an effective carrier or reservoir of infection, the host must be not only susceptible to infection but must also develop and circulate a threshold level of virus sufficient to infect the vector. Some hosts circulate less virus and are able to infect only some species of mosquitoes while others circulate more virus and can infect less efficient mosquito vectors. If the host animal develops too low a viremia to infect mosquitoes, it is considered a blind or dead-end host.

The selective feeding habits of mosquitoes must also be considered. Vectors which feed predominantly on birds, although heavily infected, may not be a hazard for mammals. A similar situation exists for vectors which feed predominantly on only certain species of mammals.

Epidemics are dependent upon the efficiency of the host species and the vector species to propagate the virus and whether both exist in sufficient numbers. The level of viral

Fig. 2.—Blood sample being taken from colt, Brownsville, Tex. Samples were sent to laboratories at Denver and at Atlanta to be tested for VEE.

activity depends not only upon the type of virus present but also on the subtype involved. These vary in their ability to infect hosts and vectors. A certain subtype may, for example, require a certain species of mosquito to maintain viral activity. However, if sufficiently high viral activity is involved other mosquitoes and additional reservoir animals could contribute to additional viral activity. Biologically, broader host and vector spectrums increase the spread and survival probabilities of arbovirus diseases. Although mosquitoes are believed to be the only important vectors of VEE, there have been observations which suggest that others should be investigated.

In the United States, in southern Florida, during the years 1963 through 1969, 99 isolations of VEE virus (an endemic strain) were made from 5 species of mosquitoes and from cotton rats. Public health officials have also reported naturally-occurring cases in humans in Florida. These were shown by serology, not by virus isolation. The strain in Florida has not caused clinical disease in horses.

Epizootic strains of VEE classified as IA

have been isolated from Colombia and Venezuela, IC in Venezuela, and IB (the one which reached Texas) has also been found in Peru, Ecuador, Central America, Mexico, and perhaps Argentina. Fortunately, the TC-83 type vaccine offers protection against all of these. Endemic strains include ID in Colombia and Panama, IE on east coast of Central America and Mexico, II Florida (only), and III and IV in Brazil. The kinetic HI test has been used to differentiate these isolates.

VEE in Mexico

Epizootic VEE had not been reported north of Panama until mid-1969 when the disease occurred in El Salvador and also invaded Guatemala, Honduras, and Nicaragua. The disease further extended into southern Mexico and into Costa Rica. As VEE progressed into Central American countries and on into Mexico, extensive use was made of the U.S. Army TC-83 vaccine in an attempt to protect individual horses and by creating an immune barrier of vaccinated animals.

Although the vaccine proved to be very effective in protecting vaccinated animals, the virus was able to breach the "immune barriers" created to contain it. A number of factors could, of course, have weakened such barriers. The most obvious possibilities are that too many animals did not receive the vaccine, in some cases the vaccine may not have been carefully cared for, or perhaps the mass vaccination program did not begin soon enough or include a large enough geographical area.

It is difficult to know just when epidemic and/or endemic VEE virus first entered Mexico. Neutralizing bodies were found there in 1962. VEE virus was isolated in the State of Veracruz in 1963. In 1966, the disease was reported as affecting some 1,000 horses (with a 30% mortality) in northern Veracruz and southern Tamaulipas states.

In August 1969, an epizootic of equine encephalitis began in the State of Chiapas adjacent to Guatemala. Approximately 300 horses died from December 1969 to February 1970. Vector control measures were undertaken in some areas. The outbreak subsided in March.

The disease again appeared in June 1970 in the same general area and an active vaccination program began in August with close to 500,000 horses being vaccinated in Chiapas, Oaxaca, and Veracruz states. The mortality rate in sick animals was about 80% with 7,000 horses reported as having died out of a population of 300,000. Deaths stopped about 7 days following vaccination; however, by the middle of September the disease had penetrated the vaccination belts to reach north of the Port of Veracruz on the Gulf area by October 1970 and north of Acapulco on the Pacific side of Mexico by November. It was estimated that during 1970, 10,000 horses died in the States of Chiapas, Oaxaca, Veracruz, Guerrero, and Michoacan. It is not known whether human deaths occurred. In April 1971, VEE manifested itself again in southern Veracruz.

During FY 1971, nearly 1 million doses of TC-83 vaccine were made available to Mexico, the most recent being in May 1971 when the 200,000 doses were sent to Mexico in anticipation of a cooperative program to vaccinate a buffer zone in Mexico along the gulf coast between Tampico, Mexico, and Brownsville, Texas. Application of the vaccine in this zone was subsidized by the United States Department of Agriculture.

However, by June, epidemic VEE virus had reached the Texas-Mexico border at Brownsville, Texas. Although initially the disease advanced mainly along the gulf coastal and other areas of lower altitude in Mexico, outbreaks also appeared inland in areas of lower rainfall in the high plains. The disease continued to advance north and west toward El Paso, Texas, and northward through the Pacific coastal and inland areas of Mexico, and continues to appear at various locations in Mexico in animals that were missed when the vaccination brigades were in the area.

Buffer zones of vaccination in Mexico also failed to hold the disease, and it advanced from southern Mexico in June 1970 to the Texas border in June 1971, a distance of 1,000 miles in a year. It spread into the United States in the last week of June.

Mexico plans to vaccinate all of her near-

ly 9 million Equidae. The task is near completion. It is difficult to obtain accurate morbidity and mortality rates. Up to 25,000 deaths of horses probably occurred in Mexico. There may have been up to 12,000 human cases with an unknown, probably very low, mortality rate.

Control of Venezuelan Equine Encephalomyelitis in the United States

This past summer's outbreak of Venezuelan equine encephalomyelitis was the number one agricultural news story of 1971 in the United States. The virus spread from Mexico into southern Texas, killed several hundred horses, and hospitalized approximately 100 people. On July 16, 1971, the U.S. Secretary of Agriculture declared the existence of a "national emergency threatening the entire horse industry." The Secretary did not have the authority to declare the emergency until the disease was confirmed in this country. This confirmation was reported on July 9, 1971.

The fight against VEE was a Herculean task. More than 4,000 practicing veterinarians, federal and State scientists and inspectors, private aerial pesticide application operators, military personnel, and chemical industry officials were battling the disease at the height of the federal-State cooperative VEE control program.

Three principal weapons were used to bring VEE under control: quarantines to prevent the movement of non-vaccinated horses from infected areas; vaccination to develop an immune horse population; and aerial pesticide spraying to reduce mosquito vector populations. Use of these weapons required massive planning and coordination.

Fig. 3.—VEE victim. This horse had wandered across the Mexican-U.S. border, was caught and held in quarantine pen, where it died.

Quarantines

Federal quarantines on all Equidae were applied to Texas on July 13, 1971, to Louisiana, Arkansas, Oklahoma, and New Mexico on July 19; and to Mississippi on August 2. Horses moving from any State under federal quarantine were required to have a veterinary health inspection and certificate and a VEE vaccination more than 14 days prior to movement. Individual states also placed restrictions on horses brought into the State, and persons wishing to move horses were advised to contact State Animal Health officials of the State concerned.

Federal quarantines were removed from New Mexico, Oklahoma, and Arkansas on September 10, 1971; from Mississippi on November 9, 1971; and from Louisiana on January 17, 1972.

Vaccination

The U.S. Department of Agriculture furnished TC-83 VEE vaccine for horses in the states of Texas, New Mexico, Oklahoma, Arkansas, Louisiana, California, Arizona, Mississippi, Alabama, Georgia, Florida, Kentucky, Tennessee, North Carolina, South Carolina, Virginia, Maryland, Delaware, and New Jersey. The Department also paid for the administration of the vaccine by veterinary practitioners in these States. More than 2.8 million horses have been vaccinated since the vaccination program began in June 1971. This broad experience with the vaccine to date indicates that it is both effective and safe for use in horses and other Equidae even though originally it was designed for use in man (people at high risk). VEE vaccine offers horses protection against VEE virus but not against eastern or western encephalomyelitis. However, vaccines have been available against these diseases for a number of years.

This organized vaccination program was not extended to other States, as they were considered to be in a lesser risk category.

However, since August 25, 1971, a licensed commercial VEE vaccine has been available in all States in which State Animal Health officials authorize its use.

Vector Control Program

Although epidemic VEE was moving rapidly northward through Mexico on rather broad fronts, it was observed that greatest disease pressures were generally along the Gulf coast area and extending inland along major water courses and in areas of lower altitudes. The vector control program in the United States was designed to reduce vector populations in more critical areas, provide time for vaccinating the horse population, and give vaccinated animals time to develop immunity. The area considered most critical was an area extending up the Rio Grande River and up the Gulf coast from Brownsville, Texas. When it was known that the disease was present in a larger area, the vector control program was extended to an area stretching from Falcon Dam, Texas, to Lake Charles, Louisiana. Unfortunately, we were unable to arrange with the government of Mexico for U.S. planes to apply the pesticide on the Mexico side of the border.

A detailed mosquito control plan outlining all aspects of the proposed treatment program was drawn up by the vector control staff of the task force sent into Texas to combat the outbreak. Cooperating federal and State agencies, the governors of Texas and Louisiana, and the President's Council on Environmental Quality reviewed and approved the plan before spraying got underway. This clearance included approval of the use of malathion, a chemical that is one of the safest insecticides in use today. Malathion residues break down a few days after application, and the formulation is relatively harmless to humans, livestock, and other warm-blooded animals. Later the Council approved the use of Dibrom, which is also nonpersistent and low in toxicity to warm-blooded animals.

Meanwhile, the Public Health Service dispatched mosquito surveillance teams to Texas because of the danger to humans who might be bitten by infected mosquitoes. The teams set up light traps throughout the southern portion of the State and began surveying mosquito species and populations. By early July, the Center for Disease Control of PHS had determined that adult mosquito counts were high enough to justify aerial spraying. Information from vector surveys enabled the vector control staff to (1) keep track of mosquito populations and buildups, (2) pinpoint areas where spraying was needed to control mosquitoes, and (3) measure the effectiveness of chemical applications.

Fig. 4.–Aerial spraying for mosquitoes in Houston area–part of vector control effort in the VEE program.

Spraying began on July 10 and when completed on August 13, 9.6 million acres had been sprayed. Local mosquito abatement agencies added another 3.1 million acres making a total of 12.7 million acres sprayed once or twice. Supplemental funding was arranged for 5 county mosquito abatement units along the coastal area of Texas.

Seven major aerial applicators received contracts. The type of aircraft to be used was restricted, for reasons of economy, speed, and proper application of the chemical, to multi-engine planes with swath widths

of at least 750 ft and airspeeds of not less than 175 mph. A single DC-3 can do the work of 8-10 single-engine spray planes. An air-ground communications network was set up connecting spray aircraft with their bases and emergency task force headquarters in Houston. Positions of all planes and acreage were plotted on a master control map in the headquarters.

As the program progressed, responsibility for compiling and evaluating survey information passed from the Public Health Service to USDA's leadership. The new mosquito survey team sent into the field was symbolic of the cooperative nature of the VEE control program, with entomologists from USDA, DOD, Texas A&M University, HEW's Center for Disease Control, and from Louisiana State University.

More than 200 federal, State, county and military personnel participated in the VEE mosquito control program. This included USDA pilots who were important factors in maintaining high spraying safety and accuracy. The pilots' daily duties included checking swath widths, making certain that only acreage scheduled for treatment was sprayed, and performing tests to maintain proper aircraft calibration and droplet size and coverage. To further insure environmental protection, pesticide monitoring teams regularly sampled water, sediment, and aquatic organisms from 19 sites throughout the area.

Cooperation received from the Chevron Chemical Company and American Cyanamid (makers of Dibrom and malathion respectively) were also important to the success of the mosquito control program. The production and movement of Dibrom and malathion were increased in coordination with program needs. In addition, both companies sent teams of experts to Texas to aid in monitoring chemical usage and safety precautions.

The VEE mosquito control program ended after 90% of the horses in the infected area and the buffer zone beyond had been vaccinated. During the program's 35 days of operation, most species of adult mosquito populations in the control area were reduced to near zero.

The low toxicity of the insecticides used,

Fig. 5.—Ground fogging for mosquitoes, Atlanta, Ga.

fine atomization and low application rates–3 oz/a for malathion and ¾ oz/a for Dibrom–enabled the task force to achieve mosquito control without endangering local ecologies. All aircraft were inspected in advance of spraying to determine that the correct nozzle sizes were used and that no leaks would cause droplet sizes in excess of those recommended. Close surveillance was maintained by USDA pilots in chase planes to assure adequate performance by spray aircraft. The insecticide flow rate was monitored in the air. Dye cards and glass slides on the ground were exposed to the spray to assure correct droplet size and dispersion.

Environmental Protection Agency personnel collected samples of water, aquatic organisms, sediment, and fish, and otherwise monitored the possible environmental effects of the spray program. Beekeepers were advised in advance to take suitable precautions.

Survey teams reported excellent control of adult *Aedes sollicitans*, *A. taeniorhynchus*, and *Psorophora confinnis*. Except where ground fogging equipment was used to spray barns and sheds, adult Anopheles were not controlled.

The role of the federal and State governments was to stop the epidemic and to minimize the number of human and equine cases rather than eradicate the disease. The control program was successful in this respect. Isolations of epidemic VEE virus were limited to south Texas and to 74 horses and 88 humans in 26 counties. No humans are known to have died from the disease.

Any discussion of the control program must necessarily make special note of the splendid cooperation that was given to the U.S. Department of Agriculture by the Department of Defense, the Department of Health, Education and Welfare, various State agencies, and a vast number of individuals. This assistance was invaluable in conducting the campaign.

Surveillance

The epidemic strain of Venezuelan equine encephalomyelitis (VEE) virus was isolated only from the southern part of Texas in

Fig. 6.–VEE vaccination program, Atlanta, Ga.

1971. It is believed that further spread of the virus in the U.S. equine population was prevented by establishing a massive barrier of vaccinated horses, and the vector control measures.

The most important part of VEE surveillance is the prompt reporting and investigation of all suspected encephalitis cases in horses or other Equidae in the entire United States. Prompt laboratory diagnostic results must be obtained from all suspicious cases to effectively control the disease. Samples from these animals will be tested for virus isolation and serum antibody to VEE, EEE, and WEE. However, it cannot be assumed that further viral spread did not or will not occur. Further, there is no information on how far the disease may have spread in those species of animals which may harbor the virus without visible evidence of disease such as small mammals.

A surveillance system is being established to further determine the area of VEE viral activity and to establish an early warning alert system regarding the spread of this disease. A primary surveillance zone has been established across the southern United States. This zone consists of a band that varies in width from 150-300 mi wide north of the United States-Mexico border, crosses through mid Texas in an east-west direction and lies north of the Gulf coast of Louisiana and Mississippi and the Alabama-Georgia-Florida borders.

In the primary surveillance zone, serum samples for antibody detection will be collected from the following animals known to be capable of developing VEE antibodies: dogs, foxes, coyotes, opossums, raccoons, deer, and other wildlife native to the area. Horses in the area will be of limited value for this purpose since most horses have been vaccinated against VEE. Hamsters and rabbits may be used as sentinel animals when necessary to provide adequate surveillance in certain strategic locations, such as in the Mississippi, Sabine, and Pecos River valleys.

To assist in this surveillance, Veterinary Services of the Animal and Plant Health Inspection Service, United States Department of Agriculture, in addition to their own personnel, is seeking assistance from other agencies. The Department of Defense will cooperate in disease and vector monitoring systems on their military bases. Also, a large number of predatory control personnel in Texas, New Mexico, Arizona, and California will assist in the surveillance. The Southeastern Cooperative Wildlife Disease Study (a study group coordinated at the University of Georgia) will participate in the surveillance in the southeastern part of the United States. In return, USDA will notify these cooperating agencies of the laboratory results for Venezuelan, eastern, and western equine encephalomyelitis.

Fig. 7.—All horses vaccinated for VEE were identified.

Summary

In the summer of 1971, VEE was brought under control using 3 principal weapons: quarantine and vaccination of horses and other equidae, and aerial pesticidal spraying for vector control.

In more than 1,500 U.S. investigations of horses suspected of having encephalitis, the

epidemic virus of VEE has been isolated from animals only in 26 south Texas counties.

The Herculean task of stopping the northward spread of VEE in the United States was accomplished only through the cooperative effort of many individuals and agencies.

A surveillance system has been established to detect any new outbreaks or other spread of VEE.